Reproductive aging patterns in primates reveal that humans are distinct

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Women rarely give birth after ∼45 y of age, and they experience the cessation of reproductive cycles, menopause, at ∼50 y of age after a fertility decline lasting almost two decades. Such reproductive senescence in mid-lifespan is an evolutionary puzzle of enduring interest because it should be inherently disadvantageous. Furthermore, comparative data on reproductive senescence from other primates, or indeed other mammals, remains relatively rare. Here we carried out a unique detailed comparative study of reproductive senescence in seven species of nonhuman primates in natural populations, using long-term, individual-based data, and compared them to a population of humans experiencing natural fertility and mortality. In four of seven primate species we found that reproductive senescence occurred before death only in a small minority of individuals. In three primate species we found evidence of reproductive senescence that accelerated throughout adulthood; however, its initial rate was much lower than mortality, so that relatively few individuals experienced reproductive senescence before death. In contrast, the human population showed the predicted and well-known pattern in which reproductive senescence occurred before death for many women and its rate accelerated throughout adulthood. These results provide strong support for the hypothesis that reproductive senescence in midlife, although apparent in natural-fertility, natural-mortality populations of humans, is generally absent in other primates living in such populations.

In human females, fertility begins to decline in the early 30s and generally reaches zero before age 50 (1–3). Reproductive senescence in women has been the subject of much discussion. Some authors describe the human pattern, in which fertility ceases in mid-lifespan, as a unique adaptation (e.g., refs. 1 and 4–6); others have described it as an artifact of the extended lifetimes that have accompanied industrialization and modern medicine in human societies (reviewed in refs. 5–9). Furthermore, some view a postreproductive lifespan as unique to humans, whereas others describe it as a common feature of many mammalian life histories (7). Some of this difference stems from whether reproductive cessation is defined as a clinical event involving follicular depletion and the cessation of sexual cycling (10), or as a life-history phenomenon in which reproductive senescence occurs at a significantly faster pace than general senescence (11). In addition, postreproductive individuals can occur in many mammalian species (7, 8), adding to the uncertainty about whether a common, extended postreproductive life, following reproductive cessation in midlife, is a distinctly human pattern. Recently, Levitis et al. (3) examined reproductive senescence in humans and in three nonhuman primate species, and concluded that the postfertile lifespan is a distinct life phase that occurs in humans but may be absent in many (but not all) other animals. The authors address the problem of how to identify a true postfertile lifespan from the occasional occurrence of postfertile individuals in a population (12). This approach also highlights the fact that to place humans precisely on the comparative landscape, data from wild populations and from multiple species of primates and other mammals are required.

Here we present such data and compare reproductive senescence patterns in seven populations of wild nonhuman primates to human patterns. Specifically, we compiled individual mortality and reproduction data for wild populations of seven species that span the Primate Order. We then modeled both mortality and reproductive senescence in humans and in these wild nonhuman primates and compared, in each species, the rate of increase in the probability of death with the rate of increase in the probability of reproductive senescence. This process enabled us to test the hypothesis that reproductive senescence in nonhuman primates, unlike in humans, occurs at the same pace as general senescence (e.g., ref. 11). The seven primate populations have been under continuous observation for a minimum of 29 y and a maximum of 50 y, and included one Madagascan prosimian (an Indriid), two New World monkeys, two Old World monkeys, and two Great Apes (13). These datasets included more than 250 combined observation-years of births and deaths on 700 individually recognized adult female primates (14) (Table S1).

We compared these primates with a human population that did not experience the reduced mortality and extended lifespan typical of industrialized or even agricultural societies. During the early and middle part of the 20th century, the Dobe !Kung population of human hunter-foragers lived in the Kalahari desert of southern Africa (Table S2). This population carried out no agriculture, subsisted on wild plants and animals, and experienced little contact with neighboring agricultural communities or medical intervention (15, 16). Human populations experiencing natural mortality and fertility, such as the !Kung, are thought to resemble those of our preagricultural ancestors in their fertility and mortality patterns (5, 17–19), making them an appropriate comparison set with wild nonhuman primates. Although human demographic parameters are variable across populations and ecological contexts, mortality parameters for the !Kung fall within the established range of values for hunter-

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Table 1. Mortality and fertility parameters for nonhuman primates and humans

Species*	Adult age interval (y)†	Modal age of mortality (y)	Modal age of fertility completion (y)	Modal age of fertility cessation (y)	Mean IBI in years (with SD)‡	Estimated age at last live birth (y)§	Postreproductive representation¶
Sifaka	6–7	23.9	26.6	22.9	1.57 (0.93)	27–28	0.02
Muriqui	8–9	42.75	Nonestimable	40.6	2.80 (0.69)	37–38	0.06
Capuchin	6–7	18.89	Nonestimable	18.0	1.89 (0.78)	24–25	0.04
Baboon	5–6	18.56	23.3	16.8	1.69 (0.46)	23–24	0.01
Blue monkey	7–8	26.14	28.2	24.6	2.30 (0.93)	30–31	0.02
Chimpanzee	14–15	38.66	Nonestimable	34.5	4.14 (2.12)	49–50	0.02
Gorilla	9–10	39.71	Nonestimable	36.5	3.48 (1.58)	40–41	0.04
Human (!Kung)	15–16	79.25	41.0	41.0	4.12 (4.00)	49–50	0.425

*Latest census date for all nonhuman primate populations in these analyses was between June 30, 2011 and October 12, 2011.
†Adult age interval is the age interval containing the mean age of first live birth in the nonhuman primate populations; for the !Kung, we used an age closer to mean age at sexual maturation; this is consistent with our use in Bronikowski et al. (14). Modal age (m) is the age at which events are centered, where the events are either death, fertility completion, or fertility cessation. Modal age uses the Gompertz parameters a and b (Table 2) as follows: $m = (1/b)\ \ln(b/a) +$ initial adult age.
‡We used mean IBI as follows: If a female lived longer than (mean IBI + 2SD) without giving birth again, we considered her to have experienced fertility completion. If a female died before reaching (Mean IBI + 2SD), we considered her fertility cessation to have been caused by her death.
§Oldest age interval in which a live birth was observed. For most of the study populations this was based on a female of estimated age.
¶Proportion of adult years lived that are postfertile (3, 12). For comparison with captive primates, Levitis et al. (3) report a postreproductive representation of 0.084 for captive *Papio hamadryas* (a taxon related to the baboons in this dataset) and a postreproductive representation of 0.224 for captive *Pan troglodytes* (chimpanzees). Fedigan and Pavelka (8) provide data on a related but different measure, the proportion of individuals that reach reproductive termination, for several other captive and free-ranging populations of primates.

forager populations, with initial mortality rates that are substantially higher and life expectancies much lower than rates for humans in developed countries (2, 20, 21).

Results

We first produced mortality tables and computed actuarial estimates of age-specific mortality hazards for females of each of the primate species [see also our earlier work (14) and *SI Materials and Methods* for a discussion of differences between this and our previous mortality analysis]. To produce comparable datasets for reproductive senescence, we measured the age at which each female in each dataset experienced her last live birth, designating each last live birth as a result of reproductive senescence or because of death. Because "reproductive senescence" can connote a gradual process of declining fertility rather than an event, we hereafter use the term "fertility completion" to denote a last live birth caused by reproductive senescence (see Table S3 for definitions of terms). A female's last live birth, at age x^*, was considered to represent fertility completion if she survived to at least age $x^* + c$ without experiencing another live birth, where c was defined as the mean plus two SDs of the interbirth interval (IBI) for that population (Table 1 and Table S3). All other last live births were because of death, except where the female was censored on the interval $x^* + c$ (i.e., was still alive at the end of our study without having experienced reproductive senescence). This process allowed us to account for the competing risk of death in measuring the completion of fertility by generating age-specific cumulative incidence functions for fertility completion (this procedure was done for each primate species but was not possible for the !Kung) (*Materials and Methods*).

Next, for each of the datasets on age of mortality and fertility completion, we tested among competing models, based on the Gompertz family of accelerating failure time models (see also ref. 14). The standard two-parameter Gompertz model provided the best fit for all of the mortality datasets, nonhuman primate and human alike. Furthermore, all of the mortality models showed significantly positive values of Gompertz parameter b, indicating that the probability of death increased with advancing age for all species (Fig. 1 and Table 2) (14).

In contrast to mortality, in four species of nonhuman primates (gorillas, chimpanzees, muriquis, and capuchins) we were unable to estimate model parameters for fertility completion; specifically, fertility completion occurred before death in only a very few individuals (Fig. 1 and Table 2). That is, only a small handful of females survived to age $x^* + c$ without either experiencing another birth or dying. For the other three nonhuman primate

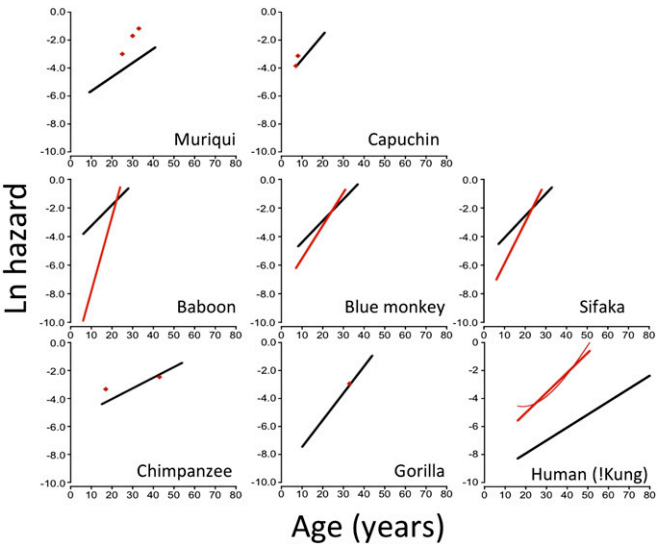

Fig. 1. Fertility completion (red symbols and lines) compared with mortality (black lines) in nonhuman primates and humans. In four primate species, so few individual experienced fertility completion before death that model parameters for fertility completion were not estimable (muriqui, capuchin, chimpanzee, gorilla); in these cases, the red diamonds indicate the age-specific cumulative incidence functions for fertility completion (adjusting for the competing risk of death). For three primate species a Gompertz model was estimable (baboon, blue monkey, sifaka); in these cases, Gompertz models of fertility completion are plotted as red lines. Note that initial rate (Gompertz a) for fertility completion was significantly lower than initial rate for mortality, so that even though fertility completion accelerated at a faster rate than mortality in these three species, relatively few individuals experienced fertility completion before death. For the !Kung population of humans, the red line showing the Gompertz model (and the thin curved red line showing the Gompertz–Makeham model, a slightly better fit) reveals that fertility completion occurs prior to death for many women in this population.

Table 2. Gompertz estimates of female mortality, fertility completion, and fertility cessation for nonhuman primates and humans

Species	Mortality models*		Fertility completion models		Fertility cessation models	
	Gompertz *a* (Initial hazard) (95% CI)	Gompertz *b* (Rate of increase in hazard) (95% CI)	Gompertz *a* (Initial hazard) (95% CI)	Gompertz *b* (Rate of increase in hazard) (95% CI)	Gompertz *a* (Initial hazard) (95% CI)	Gompertz *b* (Rate of increase in hazard) (95% CI)
Sifaka	0.0107 (0.0061, 0.0187)	0.1533 (0.1198, 0.1962)	0.0009 (0.0001, 0.0057)	0.2869 (0.1918, 0.4292)	0.0086 (0.0047, 0.0157)	0.1876 (0.1482, 0.2373)
Muriqui	0.0032 (0.0008, 0.0123)	0.10083 (0.0497, 0.2046)	Nonestimable	Nonestimable	0.0044 (0.0012, 0.0159)	0.0960 (0.0410, 0.2246)
Capuchin†	0.0201 (0.0071, 0.0567)	0.1747 (0.0747, 0.4087)	Nonestimable	Nonestimable	0.0198 (0.0067, 0.0583)	0.2003 (0.0916, 0.4377)
Baboon	0.0217 (0.0150, 0.0312)	0.1461 (0.1159, 0.1842)	0.00005 (0.1×10^{-6}, 0.0019)	0.5199 (0.3235, 0.8355)	0.0266 (0.0184, 0.0385)	0.1566 (0.1217, 0.2014)
Blue monkey	0.0091 (0.0050, 0.0168)	0.1504 (0.1156, 0.1956)	0.0020 (0.0004, 0.0095)	0.2300 (0.1460, 0.3622)	0.0099 (0.0053, 0.0183)	0.1641 (0.1260, 0.2139)
Chimpanzee	0.0121 (0.0061, 0.0241)	0.0761 (0.0485, 0.1194)	Nonestimable	Nonestimable	0.0183 (0.0096, 0.0349)	0.0563 (0.0298, 0.1064)
Gorilla	0.0006 (0.0001, 0.0028)	0.1924 (0.1417, 0.2614)	Nonestimable	Nonestimable	0.0008 (0.0002, 0.0035)	0.2054 (0.1517, 0.2780)
Human (!Kung)‡	0.0002 (0.0001, 0.0008)	0.0927 (0.0723, 0.1188)	0.0037 (0.0020, 0.0070)	0.1429 (0.1169, 0.1748)	See Fertility completion	See Fertility completion

*Model parameters differ from those in ref. 13 because only females that had experienced at least one live birth were included.
†Truncated at 20–21 for mortality analysis, which resulted in truncation at 18–19 for reproduction analyses, because of heavy censoring and small sample in later mortality age classes.
‡Data on the !Kung were collected by Nancy Howell (15) and are available in "Basic Women's Interviews" at T-Space at University of Toronto Libraries: doi:hdl.handle.net/1807/18002 (Table S2). Gompertz parameters are reported in the table for comparability with the nonhuman primates, although Gompertz-Makeham was the best fit model for human reproductive cessation (*Materials and Methods*).

species (baboons, blue monkeys, and sifaka) we were able to fit a model to the fertility completion data and, similar to mortality, the standard two-parameter Gompertz model fit the data best (Table 2). For these three primate species, the value of Gompertz *b* (rate of increase with age) was higher for fertility completion than for mortality; for two of the three species (baboons and sifaka) this difference was significant (Fig. 1, Table 2, and Table S4). However, in all three of these species the initial rate of fertility completion was significantly lower than the initial rate of mortality. As a consequence, even in these three species with measurable reproductive senescence, relatively few individuals experienced fertility completion before death. Moreover, a comparison of the modal ages of death versus fertility completion for each of these three species demonstrates that for all three, the modal age of fertility completion was centered beyond the modal age at death (Table 1; see also Table S3 for definitions) (22). This finding contrasts starkly with the Dobe !Kung, for whom modal age at fertility completion was 41.0 and modal death age was 79.25 y.

To further illustrate the fact that mortality generally eclipsed fertility completion in all seven nonhuman primates, we produced estimates of "all-cause" fertility cessation for each species, and compared these with our mortality estimates [which were all-cause mortality estimates; that is, they included mortality from all possible causes (14)]. All-cause fertility cessation was distinct from fertility completion in that it included cessation resulting from the female's death as well as cessation resulting from senescence (see Table S3 for definitions). If reproduction was terminated by death more often than by reproductive senescence, then the Gompertz models for all-cause fertility cessation should be nearly identical in Gompertz *a* and Gompertz *b* to the Gompertz models for all-cause mortality. This prediction was supported (Fig. 2 and Table 2): the overwhelming majority of fertility cessations (last live births) in the nonhuman primates were instances in which death occurred at some point after a birth, but with no evidence that the female was reproductively senescent or had completed fertility at death.

The implication of this finding is that general senescence outpaced reproductive senescence: that is, the significant acceleration in the risk of mortality with age (the positive Gompertz *b* for mortality) in all of these species means that the animals experienced somatic senescence, and the nearly superimposed models

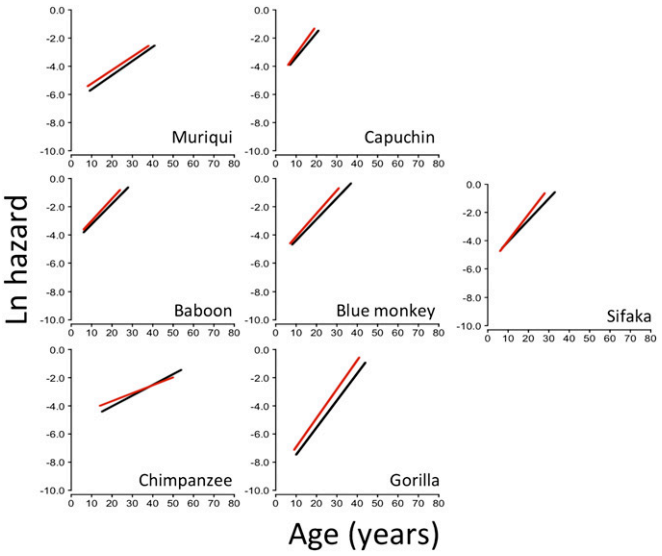

Fig. 2. All-cause fertility cessation (red) occurred at the same rate as mortality (black) for seven nonhuman primate populations. All lines represent Gompertz models. The strong similarity between the fertility cessation models and the mortality models indicates that most fertility cessation was due to death, revealing that somatic senescence eclipsed reproductive senescence in these species. Note the strong contrast with the human data in Fig. 1.

for mortality and all-cause fertility cessation indicate that in each age class somatic senescence surpassed reproductive senescence.

In this way, the nonhuman primates differed strikingly from the human population of !Kung, in which reproductive senescence was evident from the significantly higher probability, throughout adulthood, of fertility completion than of death (Fig. 1, and Tables 1 and 2) (see also refs. 2, 3, 11, 12, 21). Indeed, our data make clear that the life history pacing—irrespective of the physiological process—of reproductive senescence is different in these seven wild nonhuman primate populations than in humans. To underscore this result, we draw the reader's attention to three sets of values. First, as noted above, the modal ages at death are centered before the modal ages at fertility completion for the nonhuman primates for which this was estimable, but long after modal age of fertility completion for the !Kung (Table 1). Second, the values representing the 90th percentiles of the distributions for ages at last live birth and ages at death are plotted against each other for all species (Fig. 3) (23). The 90th percentiles of these two distributions are quite similar for the nonhuman primates, but they are quite different for the !Kung (Table S5). Finally, comparison of the postreproductive representation of adults (3, 12) in the nonhuman primates and humans is also striking: among the nonhuman primate species, 1–6% of adult-female-years are lived by postreproductive individuals, whereas in the !Kung this metric reaches 42.5% (Table 1).

Discussion

Hawkes and Smith (1) have proposed that the human postreproductive lifespan has resulted from increased longevity (decreased death rates) in the human lineage over evolutionary time without a concomitant increase in the reproductive lifespan. Our data are broadly consistent with this hypothesis; by lowering death rates but not altering the pace of reproductive senescence in the three species for which we were able to model reproductive senescence, a difference would emerge between mortality and reproductive senescence that would be qualitatively very similar to that seen in humans (Fig. 1). In other words, our data support the hypothesis that what makes humans distinct may be lower mortality that "reveals" reproductive senescence, rather than a modified pattern of reproductive senescence relative to other primates (1).

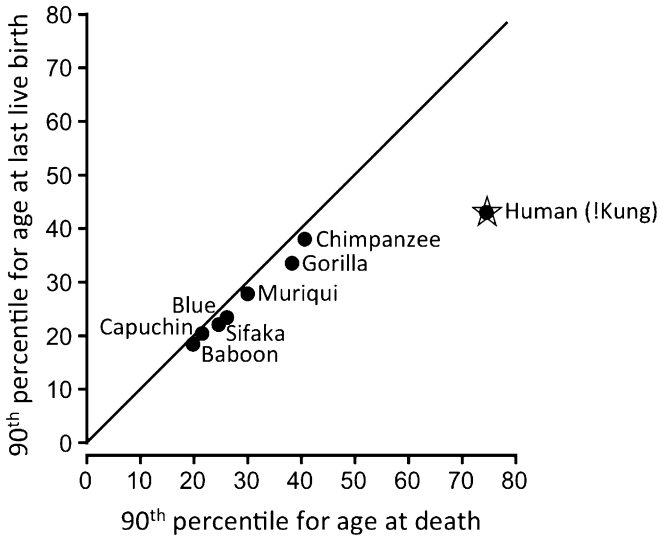

Fig. 3. The life history pacing of fertility completion is different in nonhuman primate species than in humans. The 90th percentiles of the distributions for age at death (in years) are plotted against the 90th percentiles of the distributions for age at last live birth (in years). The distributions include both censored and uncensored data. The black line represents the line of identity between the two distributions. The 90th percentiles of these two distributions are quite similar for the nonhuman primates, but they are quite different for the !Kung population of humans.

Our data also support the findings of Levitis et al. (3), whose analyses indicated that even in historical or modern human populations with little or no medical intervention, the representation of postreproductive women in the population would generally have been 40–50%, a much greater proportion than in the three nonhuman primates they examined. Here, we provide distinct information that complements and extends their results. First, by fitting failure time models for both mortality and reproductive senescence in the same individuals in multiple species, we present an explicit test of the hypothesis that reproductive senescence occurs at the same pace as mortality senescence in nonhuman primates, rather than at a faster pace as it does in humans. This approach highlights, in a unique way, the distinctiveness of the human life history. Second, by examining data on wild populations of seven nonhuman primate species, we significantly expand the comparative landscape of mammalian reproductive senescence relative to what is currently known.

Why has the human reproductive lifespan not kept pace as the human somatic lifespan has extended? Some have proposed an absolute limitation on the length of time that primary oocytes can remain viable (reviewed in ref. 8). In theory, this limitation on reproductive lifespan would constrain reproduction for any mammal whose lifespan routinely exceeded the hypothetical maximum "shelf-life" of mammalian primary oocytes (but see ref. 24). These constraints may have kept reproductive lifespan constant over human evolutionary history even as somatic lifespan increased (see also ref. 1). In support, researchers have found that an age-related decline in fertility in chimpanzees occurs in the fifth decade of life; this is similar to (although somewhat later than) the age-related decline in human fertility, despite the longer lifespan of humans even in preagricultural populations (1, 2, 21).

Relevant to this hypothesis, fertility rates for known individuals in natural populations are available from two other very long-lived mammals. Female killer whales appear to experience reproductive senescence at a faster pace than general senescence, with the consequence that killer whales, like humans, cease to reproduce by 50 y of age and have a long postreproductive life (25). In contrast, female African elephants are reported regularly to give birth into their 50s, with some births also occurring when females are estimated to be past 60 y of age (26). Studies of both species depend on estimated rather than known ages for females in the older age classes [as in most of our study populations (13, 14); see *Materials and Methods* for information on age estimation]; the reported patterns may change as known-aged animals in these study populations accumulate in the oldest age classes. Nonetheless, the striking difference between these two species based on current information indicates that the pacing of reproductive senescence relative to somatic senescence can vary a good deal across long-lived species. Furthermore, the elephant data suggest that mammalian reproductive lifespans may not experience an absolute limit, but instead may be free to evolve with increased longevity.

In contrast to explanations based on physiological constraints, adaptive explanations for the long postreproductive life of human females focus on the potential role of mothers in the survival of their offspring (27–29), or grandmothers in the survival of their grandoffspring (4, 7, 9, 18, 29–31). The "mother" hypothesis argues that the human life history is characterized by a specific set of adaptations—short IBIs, overlapping child care, and reduced reproduction late in life—that mitigate the costs of producing offspring with exceptionally long periods of dependence. Human offspring require well over a decade to achieve full nutritional independence; consequently, according to

the "mother" hypothesis, late-born infants simultaneously experience low survival themselves and put existing offspring at risk by diverting maternal resources, because they are the offspring of aged mothers (e.g., refs. 28, 29). The "grandmother" hypothesis argues that women with adult children will experience greater net fitness benefits by investing in the production and survival of grandoffspring than by continuing to reproduce themselves. Selection may act primarily via the direct benefits that grandmothers provide to grandoffspring, so that the postreproductive lifespan is a form of grandparental investment (e.g., refs. 18, 27, 30, 31). Alternatively or in addition, selection may involve intergenerational competition that is resolved in favor of younger women (4, 5). Tests of these hypotheses are beyond the scope of the present report but remain intriguing possibilities for future analyses.

Notably, each of our study populations included at least one, and usually several, postfertile females; Levitis et al. (3) cite the presence of postfertile females in a population as a necessary precondition for the evolution of a true postfertile stage as seen in humans. Levitis et al. (3) also note that a comprehensive comparative dataset, including schedules of mortality and reproduction and also detailed socioecological data on parental care and kin structure, will be needed to develop a comprehensive understanding of the contexts in which a significant postfertile lifespan may evolve. The dataset we present here is a major step in this direction.

Materials and Methods

The governments of Brazil, Costa Rica, Kenya, Madagascar, Rwanda, and Tanzania provided permission for our field studies. All research complied with guidelines in the host countries. For study-specific acknowledgments and Institutional Animal Care and Use Committee compliance, see http://demo.plhdb.org.

Measuring Mortality and Reproductive Cessation. In the nonhuman primates, we measured mortality and fertility cessation in the same individuals, including only females that lived to adulthood (i.e., to the age interval containing the mean age of first live birth) and experienced at least one live birth. First, we produced mortality tables and computed actuarial estimates of age-specific mortality hazards for females of each primate species. We next measured the age at which each female in each study species experienced her last live birth. Our analysis of fertility completion included three categories of observed last live birth. (1) A female's age at last live birth, x^*, was considered to represent fertility completion if a female survived to at least age $x^* + c$ without experiencing another live birth, where c was the species' mean IBI plus two SDs (mean IBI+2SD) (Table 1 and Table S3; see also refs. 8 and 32). (2) A female's age at last live birth occurred because of the competing risk of death if she experienced a live birth and then died before $x^* + c$. (3) A female was considered censored on the age interval x^* if she experienced a live birth and was then last seen alive before the completion of IBI+2SD; that is, if she was last seen alive before $x^* + c$.

Finally, we computed actuarial estimates of age-specific hazards for all causes of fertility cessation in these same females (including cessation resulting from the female's death) (Table S3). In computing all-cause fertility cessation, a female's age at fertility cessation was considered to be x^* if she experienced her last live birth at age x^* and died at age $x^* + a$, but her age at fertility cessation was considered censored at age x^* if she experienced her last live birth at age x^* and was last seen still alive at age $x^* + a$ (when data were last collected on her).

We followed a similar procedure for age at last live birth in the !Kung, except that we were not able to obtain fertility and mortality data on the same individuals, and the last live birth data came from interviews of living women. Consequently, we simply modeled actuarial estimates of the age-specific hazards for mortality, and for the probability of experiencing last live birth without accounting for the competing risk of death. We considered the age at last live birth to be known if the woman reported that she was past menopause, and otherwise to be censored (see *SI Materials and Methods* regarding limitations on the human comparison).

Competing Risks Analysis. We conducted a competing risks analysis of reproductive senescence (33) where the event that we were attempting to estimate was risk of last live birth because of reproductive senescence (i.e., fertility completion; see Table S3 for definitions); competing events were those in which last live birth was because of death of the female, and censored observations were last live births observed with unknown fate afterward (because observations ceased in that interval). These data on ages of events, competing events, and censored observations were analyzed with %CIF macro in program SAS (v9.3, The SAS Institute) to estimate the cumulative incidence function (i.e., the cumulative probability of reproductive senescence over time) (34).

Modeling Mortality, Fertility Completion, and Fertility Cessation. Using the distributions of ages at death and of ages at last live birth (with competing deaths removed in the analyses of primate fertility completion), we tested among competing models for acceleration in the rate of increase in each case (risk of death, risk of fertility completion, or risk of all-cause fertility cessation) based on the Gompertz family of models. The standard Gompertz model is of the form $u_x = ae^{bx}$, where u_x in this case is the age-specific probability of mortality or of experiencing end of fertility, also known as the instantaneous hazard (for mortality or end of fertility) at age x. Model selection was based on a maximum-likelihood framework implemented in program WinModest (35). We considered the two-parameter Gompertz model described above, the Gompertz model with a constant additive age-independent term (Gompertz-Makeham model), the three parameter Logistic model (i.e., Gompertz with deceleration), and the Logistic-Makeham with an additive age-independent term. The least-parameterized model (two-parameter Gompertz) is recommended unless a more parameterized model has a significantly larger likelihood. Significance testing follows a standard method of comparing twice the difference between the likelihoods of the models being compared, which is distributed as χ^2 with 1° of freedom.

The standard Gompertz model provided the best fit for all mortality datasets, nonhuman primate and human alike, and the best fit for the three nonhuman primate datasets for which a fertility completion model was estimable (Table S6). This model also provided the best fit for all of the all-cause fertility cessation datasets. Consequently, we examined two parameters from the Gompertz models that, together, describe aging dynamics within each population (22, 36). The first metric was the initial rate, represented by the Gompertz parameter a, of mortality, fertility completion, or fertility cessation at the onset of adulthood. The second metric was the rate of increase in the hazard for the event, Gompertz parameter b (i.e., the rate of increase in age-specific mortality, age-specific fertility completion, or age-specific fertility cessation with advancing adult age). For fertility completion in the !Kung, the Gompertz-Makeham model provided the best fit (Table S6); here we present the Gompertz-Makeham model for this dataset as well as the Gompertz model, the latter for comparability to the nonhuman primate datasets.

Age Estimation for the Nonhuman Primates. For most individuals in each study population, individual ages were known to within a fraction of a year. Exceptions occurred for individuals that were present at the initiation of each study or that immigrated into the study population during the course of the study. Ages for these animals were estimated based on known patterns of maturation and age-related changes in physical characteristics in these populations. We conducted a sensitivity analysis of the impact of uncertainty in age estimates for this small fraction of animals with estimated age. In general, this uncertainty manifests in the assignment of a species-specific maximum lifespan, which is not a focus of this report. Our conclusions are therefore robust to the uncertainty in magnitude of aging rate, young-to-middle–aged adult mortality estimates, and overall shape of survival curves (see also ref. 14).

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